NEURO-ISCHEMIC ULCERS

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NEURO-ISCHEMIC ULCER UNDER HALLUX

A 68-year-old obese male patient with type 2 diabetes diagnosed at the age of 46 years visited the outpatient diabetic foot clinic because of two chronic ulcers on his right hallux. He was treated with a combination of sulfonylurea during the day and a mixture of 20% rapid acting–80% intermediate acting insulin before dinner; he also had dislipidemia which was being treated with simvastatin.

On examination, he had severe diabetic neuropathy (no sensation of light touch, pin prick, temperature, 5.07 monofilament, absence of Achilles tendon reflexes and a vibration perception threshold over 50 V).

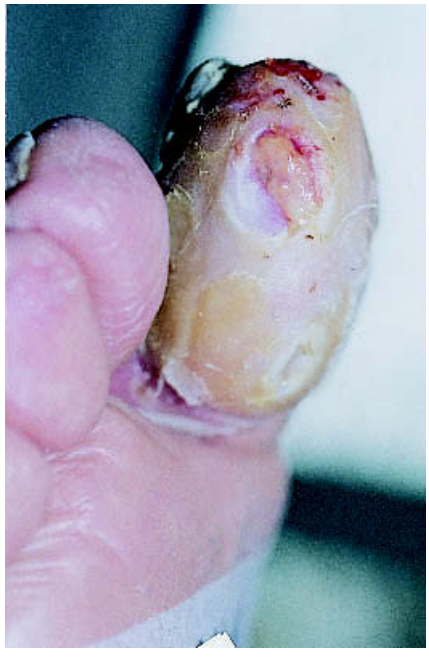

Figure 6.1 Infected full thickness neuro-ischemic ulcers under the right hallux with edema and superficial ulceration at the tip and a subungual hematoma caused by inappropriate footwear

Peripheral pulses were palpable, but the ankle brachial index was 0.7 bilaterally.

Two painful full thickness neuro-ischemic ulcers with sloughy bed were seen under his right hallux; edema and superficial ulceration at the tip of his hallux, and subungual hematomata of the first two toes were also present (Figure 6.1). There was no X-ray evidence of osteomyelitis. Sharp debridement was carried out and he was treated empirically with amoxicillin–clavulanic acid. No pathogen was isolated on swab cultures, probably due to the use of local antiseptics. Offloading of pressure was successful with the help of appropriate therapeutic footwear (see Figure 3.36) and the ulcer began to heal smoothly (Figure 6.2); 3 months after the initial visit it had healed completely (Figure 6.3).

Keywords: Neuro-ischemic ulcer

NEURO-ISCHEMIC ULCER WITH OSTEOMYELITIS UNDER THE HALLUX

A 67-year-old man who had type 2 diabetes since the age of 44 years and was being treated with insulin, visited the diabetic foot clinic because of an ulcer on his left hallux. He had acceptable diabetes control. He had proliferative retinopathy which had been treated with laser, and intermittent claudication at 400 m. He had smoked for 27 years.

Twenty days before his visit, he had worn a new pair of shoes and driven his car for a long distance. The following day a blister developed on his left great toe. Within a day the area became edematous and black.

On examination the patient was found to have a deep, foul-smelling ulcer with

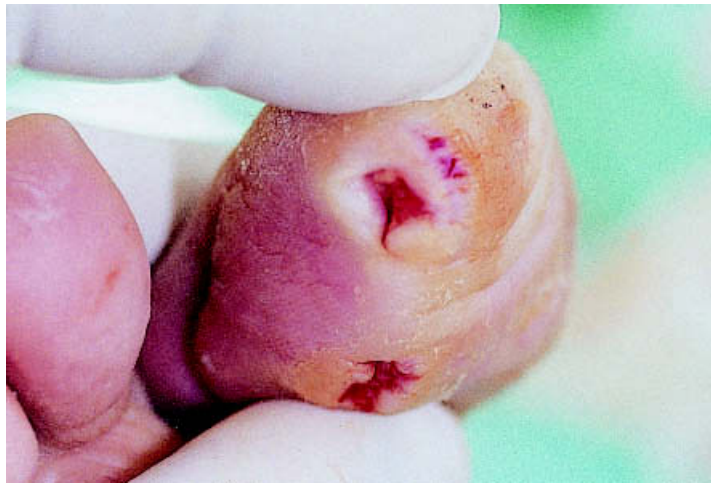

Figure 6.2 Healing of the ulcers shown in Figure 6.1

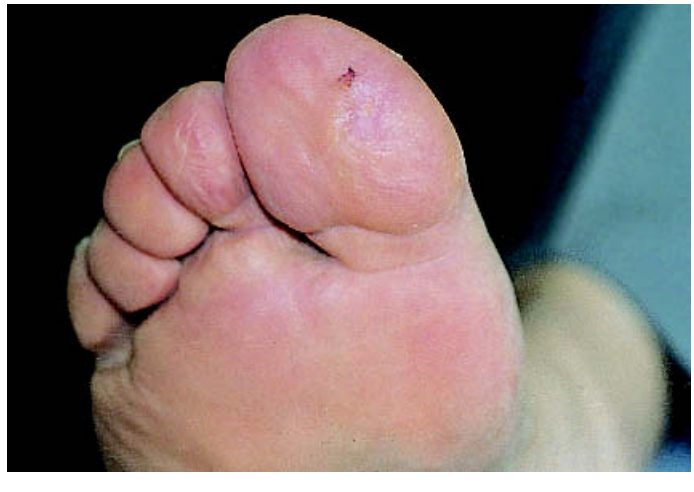

Figure 6.3 Hallux ulcers shown in Figures 6.1 and 6.2 after they have fully healed

gangrenous areas and a purulent discharge over the medial and dorsal aspect of his left great toe (Figure 6.4).

Peripheral pulses were weak and the ankle brachial index was 0.8. The patient could not feel pain, temperature, light touch, or a 5.07 monofilament. The vibration perception threshold was 40 and 45 V at the tips of the left and right great toe respectively.

Sharp debridement was carried out revealing the underlying bone. An X-ray showed osteomyelitis of the distal phalanx (Figure 6.5). The patient was treated empirically with ciprofloxacin and clindamycin as a swab culture from the base of the ulcer had revealed *Staphylococcus aureus*. Despite local foot care and the systemic antibiotics, the ulcer was still active and osteomyelitis spread locally affecting

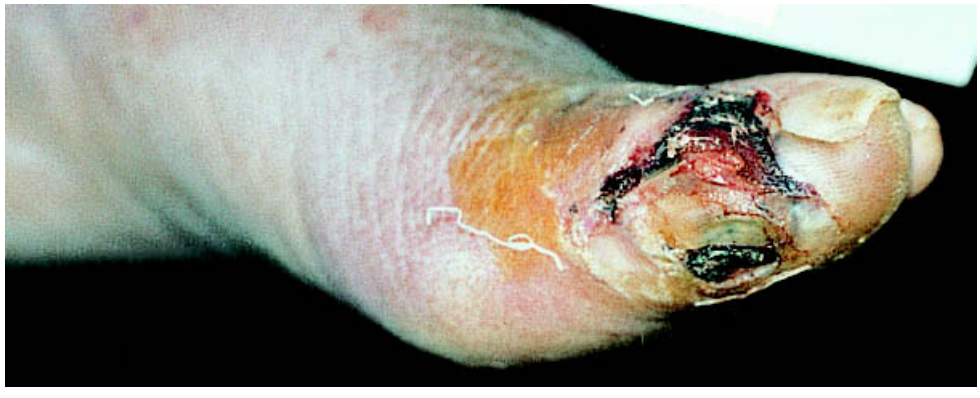

Figure 6.4 Deep tissue infected neuro-ischemic ulcer with necrotic areas on the left hallux

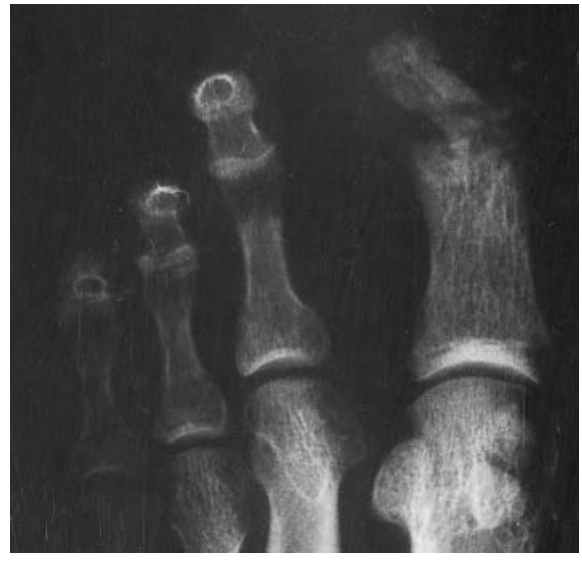

Figure 6.5 Plain radiograph of the foot illustrated in Figure 6.4, showing osteomyelitis involving proximal interphalangeal joint and adjacent phalanges of the left hallux

the proximal phalanx. Three months after his first visit the patient's left great toe was amputated (Figure 6.6). Antimicrobial treatment was continued for 2 weeks after the amputation.

The consequences of hallux disarticulation at the metatarsophalangeal joints have been discussed previously (see Figure 3.32).

Keywords: Neuro-ischemic ulcer; osteomyelitis; amputation

NEURO-ISCHEMIC ULCER ON THE DORSUM OF THE FOOT

A large (3.5 × 2.0 cm) painless neuro-ischemic ulcer developed on the right foot of a 68-year-old male patient with type 2 diabetes which had been diagnosed at the age of 61 years. Peripheral pulses were

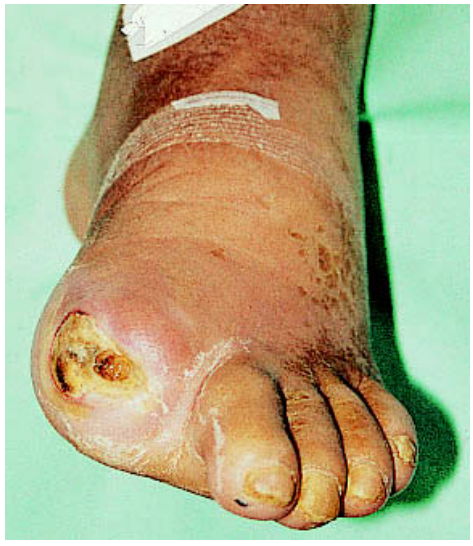

Figure 6.6 Disarticulation at the metatarsophalangeal joint of left hallux of the patient whose foot is shown in Figures 6.4 and 6.5

weak and the ankle brachial index was 0.6. At the base of the ulcer the fascia of the dorsum of the forefoot was exposed (Figure 6.7). There were no signs of infection. A subungual hematoma of the hallux and an ulcer which was healing on the second toe were noted in addition to significant ankle edema. The ulcer was a result of friction between the foot and the forepart of the patient's narrow shoe upper (vamp) following the rupture of a large blister.

Therapeutic footwear was prescribed (Figure 6.8) and the ulcer healed within 2 months.

Appropriate footwear was prescribed subsequently in an attempt to avoid recurrence of the ulcer (preventive footwear). Such footwear is made of soft, self-moldable material without any seams, and has extra depth in order to accommodate an

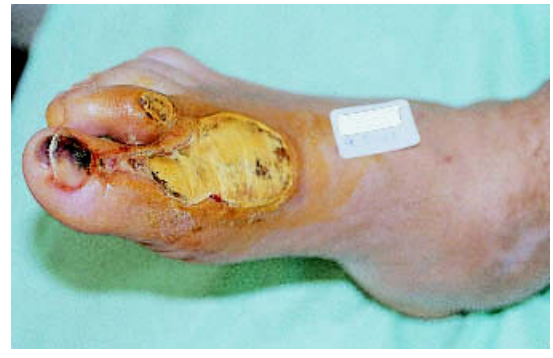

Figure 6.7 A large neuro-ischemic ulcer with exposed fascia on the dorsum of the forefoot. There is a subungual hematoma of the hallux and a healing ulcer on the second toe in addition to significant ankle edema

Figure 6.8 Therapeutic footwear for ulcers on the dorsum of the forefoot

appropriate insole and the forefoot deformities (Figure 6.9). Skin injuries due to shoe friction are thus avoided; this is essential for patients whose skin is thin and fragile due to arterial disease.

Keywords: Foot; dorsum; neuro-ischemic ulcer

INTERDIGITAL NEURO-ISCHEMIC ULCER

A neuro-ischemic ulcer on the lateral aspect of the fourth toe (Figure 6.10) was caused by pressure from the patient's little toe. Shoes with narrow toe boxes are often the cause of such ulcers. Mild callus formation — due to pressure of the adjacent toes — was seen around the ulcer. The patient suffered mild discomfort. A silicone-ring (Figure 6.11) was used to keep the third and fourth toes apart until the ulcer healed. The patient was instructed in foot care and the correct footwear was also prescribed.

Keywords: Interdigital neuro-ischemic ulcer

Figure 6.9 Preventive footwear and shock-absorbing insole for patients at risk for ulceration. The shoe upper is made of soft self-moldable material without seams. A high toe box facilitates insertion of the insole

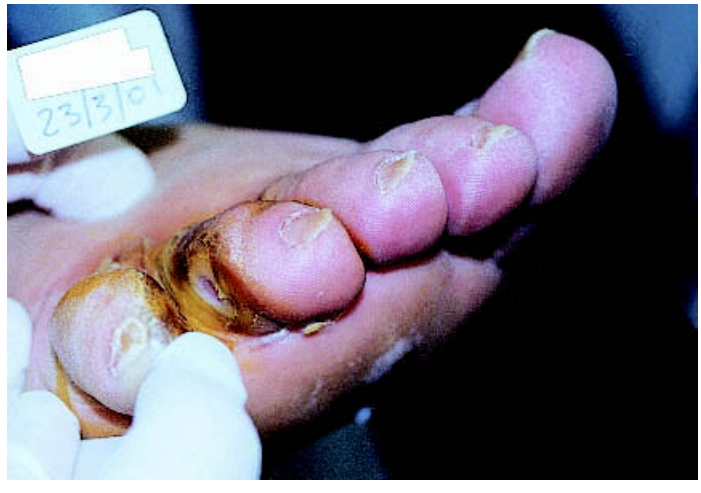

Figure 6.10 Interdigital neuro-ischemic ulceration caused by tight shoes

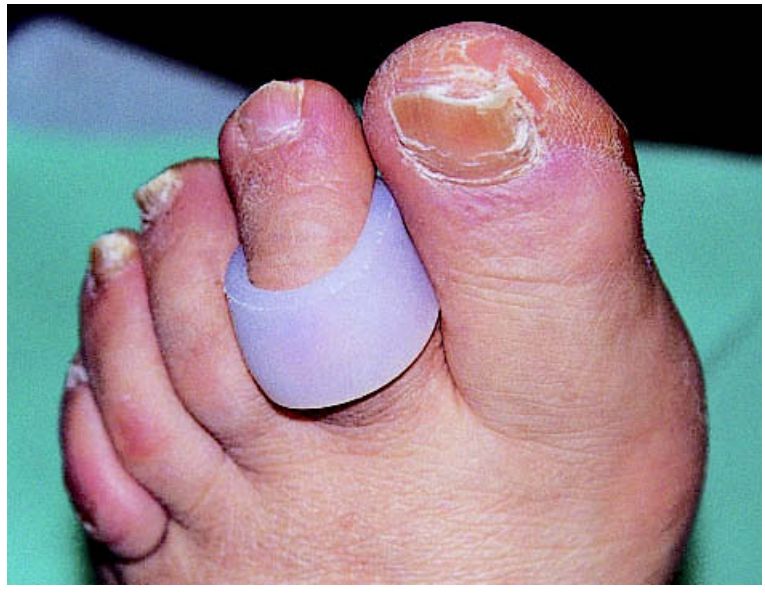

Figure 6.11 Silicone ring used to keep adjacent toes apart

NEURO-ISCHEMIC ULCERS ON THE MEDIAL SIDE OF THE FOOT

A 51-year-old obese male patient who had type 2 diabetes since the age of 34 years and was currently being treated with glimepiride, visited the outpatient diabetic foot clinic. During the past 12 months his diabetes control varied (HbA_{1c}: 7.5–9.0%). The patient had hypertension which was being treated with quinapril and furosemide; he was a smoker until the age of 49 years and was currently being treated with inhalations of ipratropium bromide and oral theophylline for the management of chronic obstructive pulmonary disease. Painful leg and foot neuropathy was treated with carbamazepine, with fair results. A vascular surgeon had prescribed low dose aspirin and buflomedil. Background retinopathy (hemorrhages and soft exudates) and nephropathy were also diagnosed.

Moderate pes planus was noted (Figure 6.12). Reflexes of both knees and Achilles tendons were absent, and there was decreased deep and superficial sensation (light touch, cold and warm sensation, monofilament, pin-prick sensation and vibration perception threshold). The patient also suffered from venous insufficiency, mild ankle edema and skin atrophy. Hematocrit, 35.5%; creatinine, 1.3 mg/dl (114.9 μmol/L); urine protein, 1100 mg/24 h; normal plasma lipid profile; BMI, 32 kg/m^2. A 4-cm aneurysm of the abdominal aorta was found by ultrasound scan. The ankle brachial index was 1.4 due to calcification of the posterior tibial and the dorsal pedal arteries. A triplex of the leg arteries

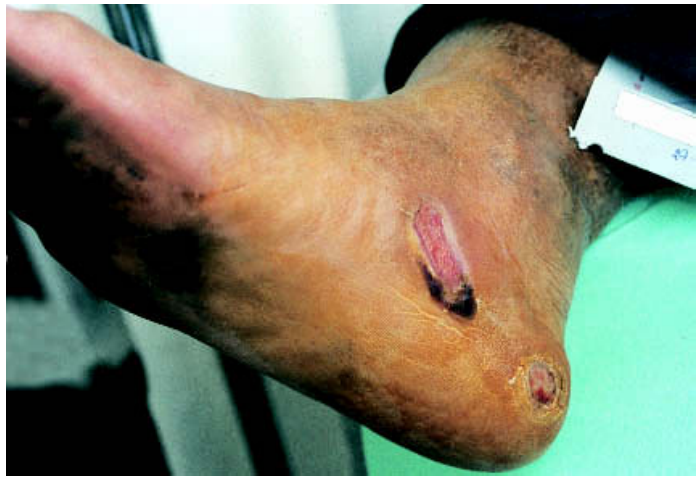

Figure 6.12 Neuro-ischemic ulcers on the hindfoot

revealed significant diffuse stenoses mainly of the arteries in the left leg.

The patient had two painful superficial ulcers on the medial aspect of his right foot due to trauma from his footwear, which he first noticed 3 months earlier. He used topical povidone iodide with no improvement.

The ulcers were clean without signs of infection. A mild callus had formed as a result of shoe friction. At the clinic the ulcers were debrided on a weekly basis and dressed with standard gauge with 15% saline. They healed completely in 1 month. Povidone iodide was discontinued as it impairs wound healing. Instruction in appropriate foot care and foot hygiene was provided, and suitable footwear was prescribed.

Neuro-ischemic ulcers comprise almost 40% of all diabetic foot ulcers. Ischemic ulcers develop at sites which are not stressed by high pressure, such as the lateral, medial or dorsal aspect of the foot and are usually painful. Intervention with vascular surgery (bypass grafting or percutaneous transluminal angioplasty) is usually needed in order to restore the blood supply to the periphery.

Keywords: Peripheral vascular disease; neuro-ischemic foot ulcers; pes planus

NEURO-ISCHEMIC ULCER ON THE FIRST METATARSAL WITH OSTEOMYELITIS

An ostensibly small, painless neuro-ischemic ulcer on the medial-plantar area of the first metatarsal head with callus formation and purulent discharge was the reason for this patient's visit (Figure 6.13). Claw deformity of lesser toes was present. After debridement, a $1.5 \times 1.0 \times 1.0$ cm ulcer was revealed. A plain radiograph showed osteomyelitis of the first metatarsal head. *Staphylococcus aureus* was isolated from

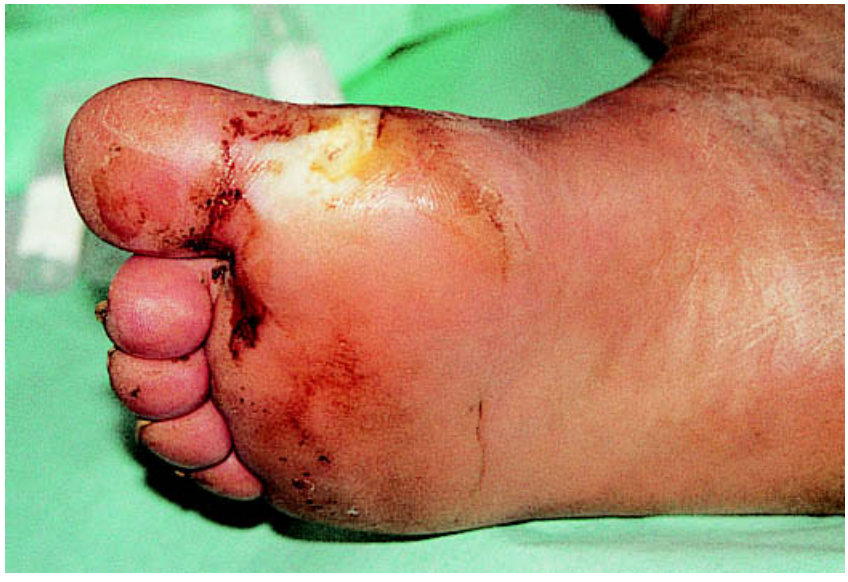

Figure 6.13 An ostensibly small neuro-ischemic ulcer complicated by osteomyelitis. Claw deformity of lesser toes is also apparent

the discharge and the patient was treated with clindamycin for 6 months, with a good outcome.

Keywords: Neuro-ischemic ulcer; osteomyelitis

NEURO-ISCHEMIC ULCERS ON THE MIDSOLE AND HEEL

After surgical debridement this diabetic patient suffered from two painful neuro-ischemic ulcers on the right midsole and the medial aspect of the heel (Figures 6.14 and 6.15). Cellulitis around the plantar ulcer was observed. Pedal pulses were weak and the ankle brachial index was 0.7. The ulcers resulted from ruptured blisters which had developed after prolonged walking in new shoes. Initially the ulcers were painless due to peripheral neuropathy, and the patient continued his activities. An angiogram showed mild atheromatous disease at the iliac and common femoral artery, severe stenosis in the middle of the right superficial femoral artery and a lesser degree of stenosis in the popliteal arteries. Balloon angioplasty of the right superficial femoral artery was carried out and an intravascular stent was inserted. Use of a wheelchair to offload pressure, adequate use of various antibiotics and a revascularization procedure resulted in complete healing of the ulcers.

Keywords: Neuro-ischemic ulcers

NEURO-ISCHEMIC ULCER ON THE FOREFOOT WITH OSTEOMYELITIS

A 64-year-old male patient with type 2 diabetes that had been diagnosed at the age

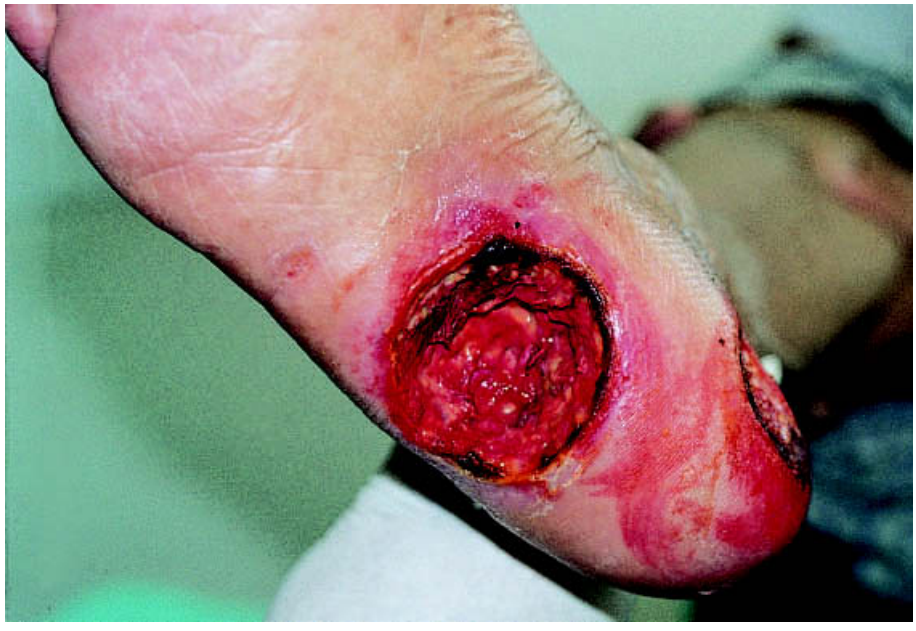

Figure 6.14 The deep neuro-ischemic ulcer with surrounding cellulitis on the right sole resulted from ruptured blisters which developed after prolonged walking in new shoes

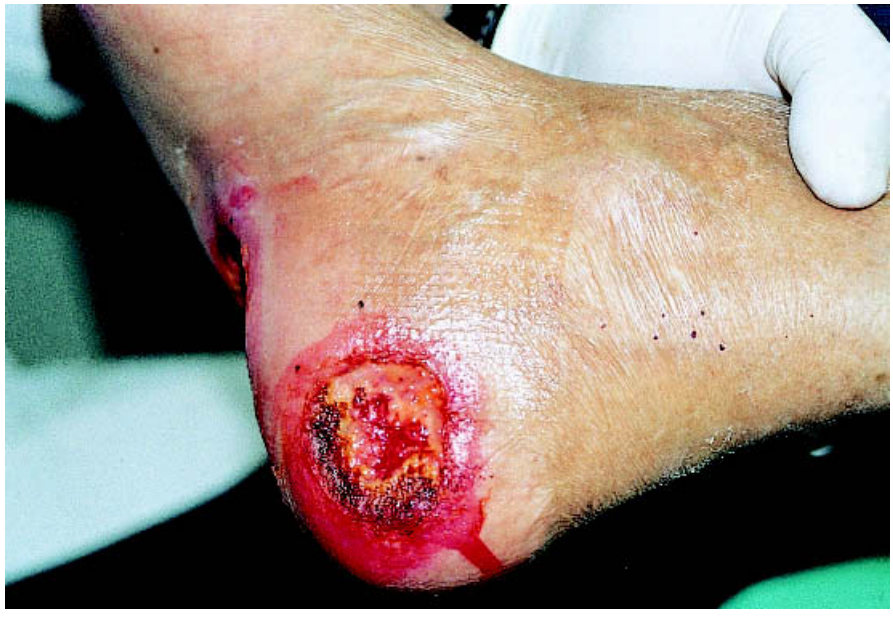

Figure 6.15 Heel ulcer in the patient whose foot is shown in Figure 6.14. The yellowish appearance of the bed of the ulcer is indicative of ischemia

of 55 years, was referred to the outpatient diabetic foot clinic because of an infected chronic ulcer on his left foot. The patient had a history of heart failure, ischemic heart disease and stage II peripheral vascular disease (intermittent claudication) according to the Fontaine classification (see Chapter 1). He also reported burning pain and numbness in his feet which worsened during the night. Three months earlier, after a long walk, the patient noticed the appearance of a small ulcer under his left first metatarsal head. He did not ask for medical help at that time since he felt no pain. A yellowish discharge was present on his socks and the insole of the left shoe.

On examination, an infected, foul-smelling ulcer was observed under his second metatarsal head, extending into the second web space (Figure 6.16). Another ulcer surrounded by callus was also noted under the first metatarsal head. Peripheral pulses were weak on both feet. He had findings of severe diabetic neuropathy. After debridement a purulent discharge emanated from the deeper tissues of the dorsum of the foot. A plain radiograph did not reveal osteomyelitis. A culture of the pus revealed *Staphylocccus aureus*. The patient was afebrile, but he was admitted to the hospital and treated with i.v. administration of amoxicillin–clavulanic acid. Two weeks after his admission osteomyelitis at the proximal phalanx of the second toe was diagnosed. The patient sustained a second toe disarticulation at the metatarsophalangeal joint. The wound healed well, and the infection subsided completely.

Several relapses of foot ulceration occurred in the following years. The patient attended the foot clinic erratically and did not wear appropriate footwear. Two years after his amputation a new neuro-ischemic ulcer developed on the midsole (Figure 6.17) caused by a worn-out insole.

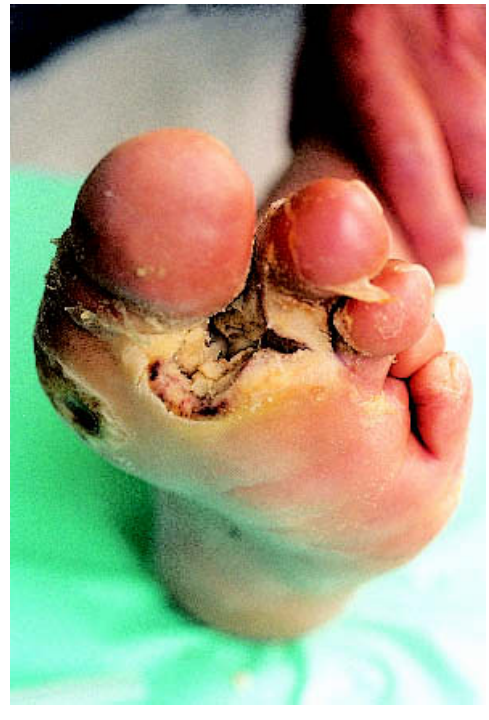

Figure 6.16 An infected neuro-ischemic ulcer soaked in profound discharge, on the plantar area between the first and the second left metatarsal heads extending into the second web space. A second ulcer surrounded by callus is also seen under the first metatarsal head

A new neuro-ischemic ulcer under his first metatarsal head was also present. There was callus formation below his disarticulated second toe.

Refusal to wear suitable footwear is a major problem in patients at risk for foot ulcers. Although there is evidence to suggest that the correct footwear reduces the incidence of foot ulcers, and many healthcare systems cover 70–100% of the cost of preventive footwear (shoes and insoles), only 20% of patients wear appropriate footwear on a regular basis. Effective education may increase this rate. In addition, the recurrence of ulcers after initial healing is also common. A recurrent ulcer is

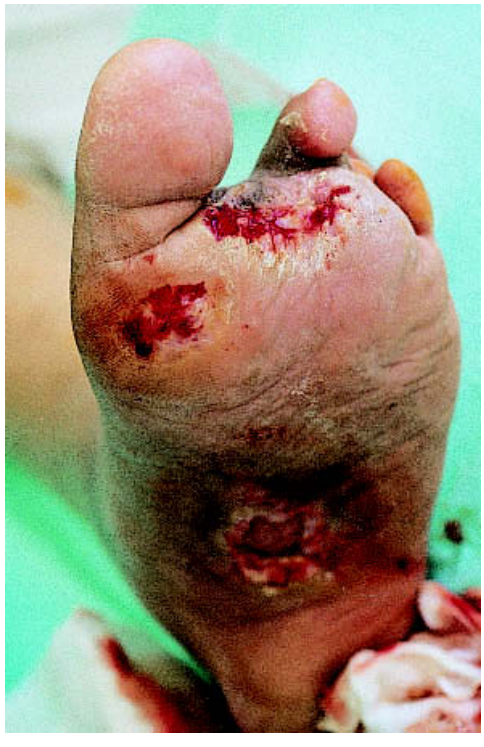

Figure 6.17 The same patient whose foot is illustrated in Figure 6.16, two years after second toe disarticulation. A neuro-ischemic ulcer caused by a worn-out insole is seen on midsole. A recurrent neuro-ischemic ulcer is present under the first metatarsal head. A callus has formed below the disarticulated second toe

defined as any tissue breakdown at the same site as the initial ulcer occurring during the 30 days following the initial healing. Any new ulcer that occurs at the same site within 30 days of healing is considered to be part of the original episode. An ulcer at a different site is considered to be a new episode independent of the time of its development. New ulcers develop at the same or different sites in a foot with prior foot ulceration in about 50% over 2–5 years. Thus the healing of an ulcer is just the first step in the management of the patient at risk. Appropriate education, prescription of the correct footwear and reduction — if possible — of the risk factors for foot ulceration (correction of foot deformities, regular callus removal, improvement in vascular supply to the feet), may reduce the risk for recurrence of foot problems in patients with diabetes.

Keywords: Neuro-ischemic ulcer; recurrent ulcers; compliance with suitable footwear

NEURO-ISCHEMIC ULCER ON THE HALLUX WITH OSTEOMYELITIS

A 76-year-old female patient with type 2 diabetes diagnosed at the age of 62 years, attended the outpatient diabetic foot clinic for a chronic ulcer on the right hallux. She had a history of ischemic heart disease and peripheral vascular disease.

On examination she had findings of peripheral neuropathy. Pedal pulses were weak on both feet. The patient had a painful neuro-ischemic ulcer with dimensions $1.0 \times 1.0 \times 0.4$ cm and a sloughy base on the medial aspect of the right hallux caused by a tight shoe (Figure 6.18). A plain radiograph revealed osteomyelitis involving the condyle of the proximal phalanx of the hallux (Figure 6.19). The ankle brachial index was 0.6. Duplex ultrasonography of the arteries of the legs revealed multilevel bilateral atherosclerotic disease in her superficial femoral arteries and severe stenosis in the arteries of her left tibia. The pedal arteries were not involved. The patient underwent a femoropopliteal and a popliteal-peripheral bypass. Since sharp debridement of the ulcer was too painful, a dextranomer was applied for mechanical debridement on a daily basis. A swab culture and a culture of

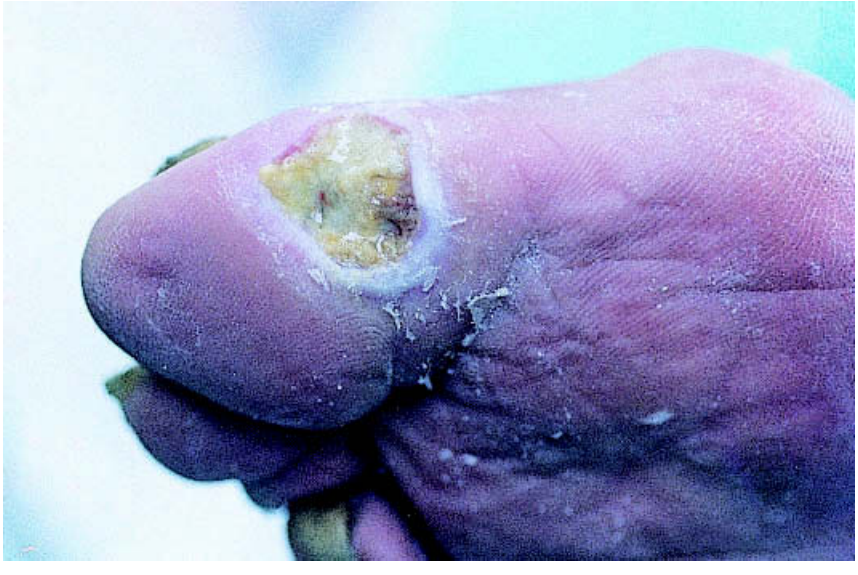

Figure 6.18 Neuro-ischemic ulcer with a sloughy base on the medial aspect of the right hallux

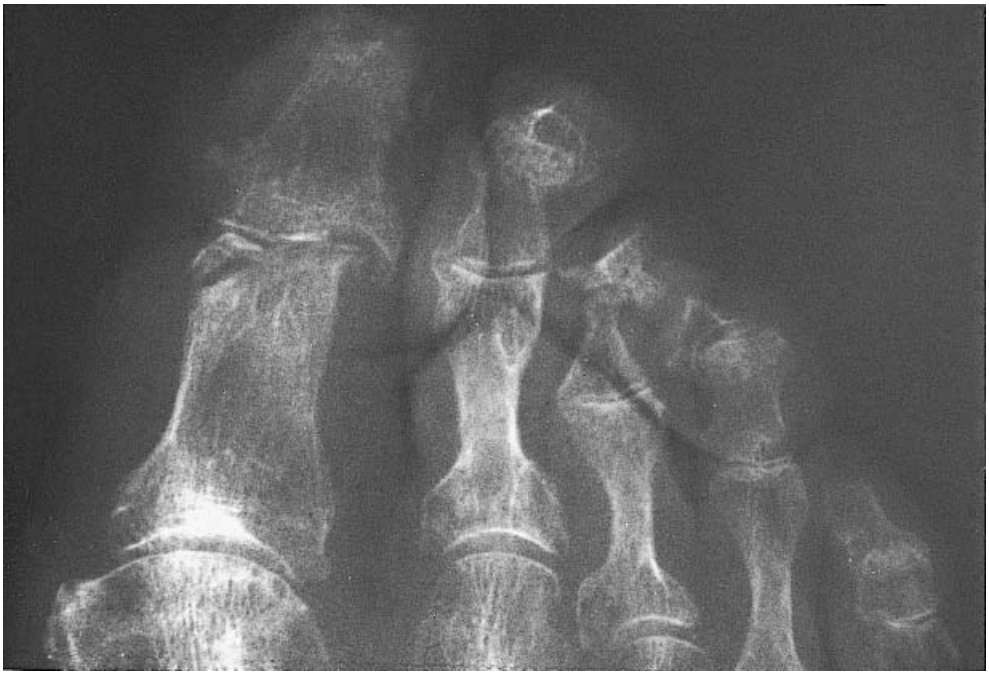

Figure 6.19 Osteomyelitis of the condyle in the proximal phalanx of the hallux of the foot shown in Figure 6.18

the sequestrum seen in a plain radiograph revealed *Pseudomonas aeruginosa* and the patient was treated with ciprofloxacin. With local wound care and antibiotic treatment the ulcer healed completely in 12 weeks (Figure 6.20). She continued with antibiotic treatment for a total of 6 months.

Inadequate blood supply prevents healing of foot ulcers especially when they are complicated by osteomyelitis.

Debridement of an ulcer is the cornerstone of the management of active, acute or chronic wounds. The aim of debridement is to remove fibrin (white, yellow or green tissue seen on the bed of an ulcer) and necrotic tissue (black tissue) and to produce a clean, well vascularized wound bed. Types of debridement are as follows:

- Sharp surgical (using scalpels), the gold standard for wound preparation, removes both necrotic tissue and microorganisms
- Mechanical (using wet-to-dry dressings, hydrotherapy, wound irrigation and dextranomers)
- Enzymatic (using chemical enzymes such as collagenase, papain or trypsin in a cream or ointment base)
- Autolytic debridement (using *in vivo* enzymes which self-digest devitalized tissue such as hydrocolloids, hydrogels, and transparent films)

Callus formation at the borders of neuropathic ulcers should be removed. The majority of patients with severe diabetic neuropathy feel no pain, therefore extensive sharp debridement or even operations on the feet can be performed without anesthesia.

The use of enzymatic debridement is increasing. Chronic wounds are enzymatically debrided in elderly patients when regular, sharp debridement is not possible, e.g. if the necrotic zone is thin; in ulcers with sinuses; and as an additional procedure to sharp debridement. Combination of collagenase with hygrogels or alginates seems to have synergistic effects.

Autolytic debridement uses the body's own enzyme and moisture to re-hydrate, soften and finally liquefy hard eschar and slough. It is selective, as only the necrotic tissue is liquefied, and painless to the patient. Its main indication is non-infected ulcers with mild to moderate exudates. Autolytic debridement can be achieved with the use of occlusive or semi-occlusive dressings which maintain the wound fluid in contact with the necrotic tissue. (For a more detailed description of the different types of dressings and their indications see Chapter 2.)

The use of sterile maggots (biosurgery, larval therapy, maggot debridement

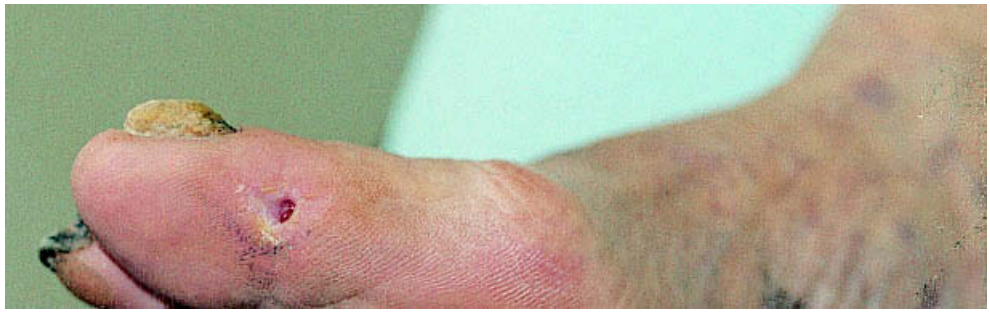

Figure 6.20 The final stages of ulcer healing in the foot shown in Figures 6.18 and 6.19. Note the chronic onychomycosis of the hallux with brown discoloration and thickening of the nail

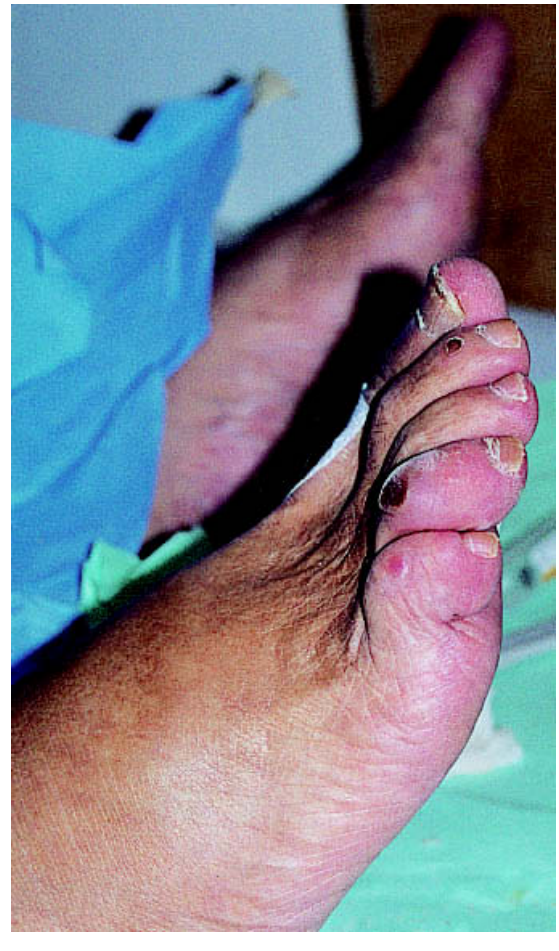

Figure 6.21 Neuro-ischemic ulcers on the dorsum of claw toes

Figure 6.22 Commercially-available preventive footwear with high toe box and minimal seaming for forefoot deformities

therapy) is a practical and highly cost-effective alternative to conventional dressings or surgical intervention in the treatment of sloughy or necrotic wounds. It is also a valuable tool in cases where wounds have been infected with antibiotic-resistant pathogens.

All chronic wounds are contaminated with bacteria. Studies have shown that a burden of 1.0×10^6 colony-forming units per gram of tissue can cause significant tissue damage and impair healing. The use of cadexomer iodide decreases microbial load, and is particularly useful in the treatment of wounds colonized by methicillin-resistant *Staphylococcus aureus*, *Pseudomonas aeruginosa* or *Candida albicans*.

Other local antimicrobials are also effective against a wide range of common microorganisms.

Keywords: Neuro-ischemic ulcer; osteomyelitis; types of debridement

NEURO-ISCHEMIC ULCERS ON THE DORSUM OF CLAW TOES

Severe claw toe deformity, combined with peripheral diabetic neuropathy and vascular disease, predisposes to ulceration of the dorsum of the toes after repetitive trauma due to irritation of the thin skin by inappropriate shoes (Figure 6.21). The use of extra depth shoes such as those shown in Figure 6.22, in addition to basic foot care, should be sufficient to ensure ulcer healing and prevention of recurrence, provided the ulcers are not infected Non-invasive vascular testing of this patient revealed multilevel stenosis of the arteries in both legs. The patient was referred to the vascular surgery department.

Keywords: Neuro-ischemic ulcers on the dorsum of toes; preventive footwear; claw toes

NEURO-ISCHEMIC ULCER WITH OSTEOMYELITIS OVER THE FIFTH METATARSAL HEAD

A 49-year-old male patient with a 4-year history of type 2 diabetes being treated with gliclazide, and an 8-year history of multiple sclerosis, was admitted because of mild fever and ulcers on his right foot. He had sustained an amputation of the last two phalanges of his right fifth toe 2 years before admission.

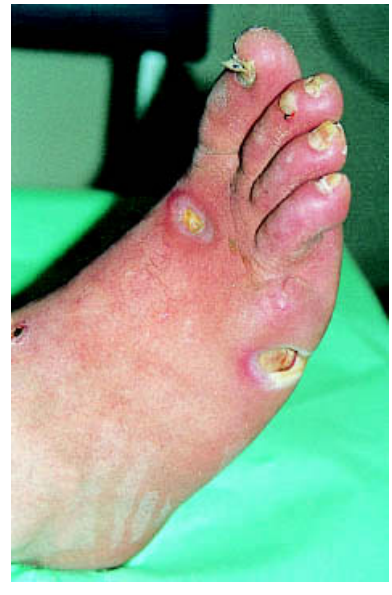

Figure 6.23 Neuro-ischemic ulcers on the right foot over the fifth and first metatarsal heads. The last two phalanges of the fifth toe have been amputated and there is a superficial ulcer on the dorsum of the second toe. Onychodystrophy is due to peripheral vascular disease

On examination he had a temperature of 37.9 °C, a pulse rate of 82 pulses per minute and his blood pressure was 140/80 mmHg. An infected ulcer was present on the upper aspect of his foot over the base of his amputated toe, and a second one over the plantar aspect of the fifth metatarsal head (Figure 6.23). He had hypoesthesia in both feet, and absence of pulses in his right leg and foot. There were pulses in his left foot and both femoral arteries. Achilles tendon reflexes were reduced and he had a Babinski sign on the right foot. His white blood cell count was 12,200/mm^3 with 74.7% neutrophils. His erythrocyte sedimentation rate was 38 mm/h. Blood glucose was 188 mg/dl (10.4 mmol/l) and his HbA_{1c} was 7.5%. Protein was present in a urine sample. An X-ray revealed osteomyelitis of the head of the fifth metatarsal, right under the ulcerated area (Figure 6.24). The patient was treated empirically with clotrimoxazole and clindamycin. *Strenotropomonas maltophilia* was isolated from a swab culture

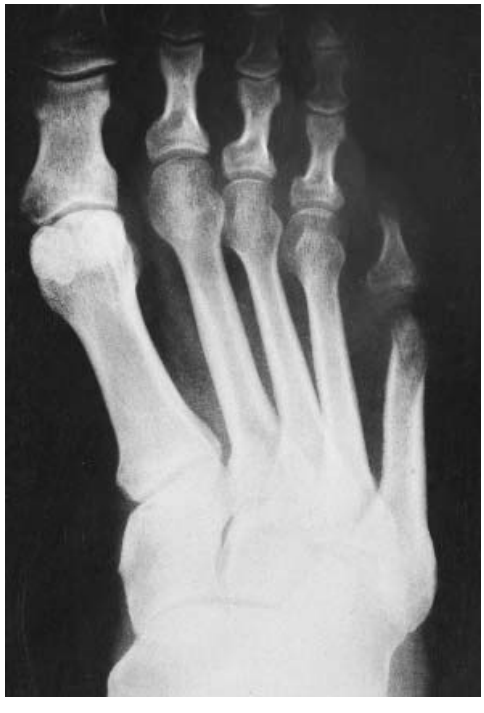

Figure 6.24 X-ray of the foot shown in Figure 6.23. There is osteomyelitis in the fifth metatarsal head and the distal phalanges of the fifth toe have been amputated

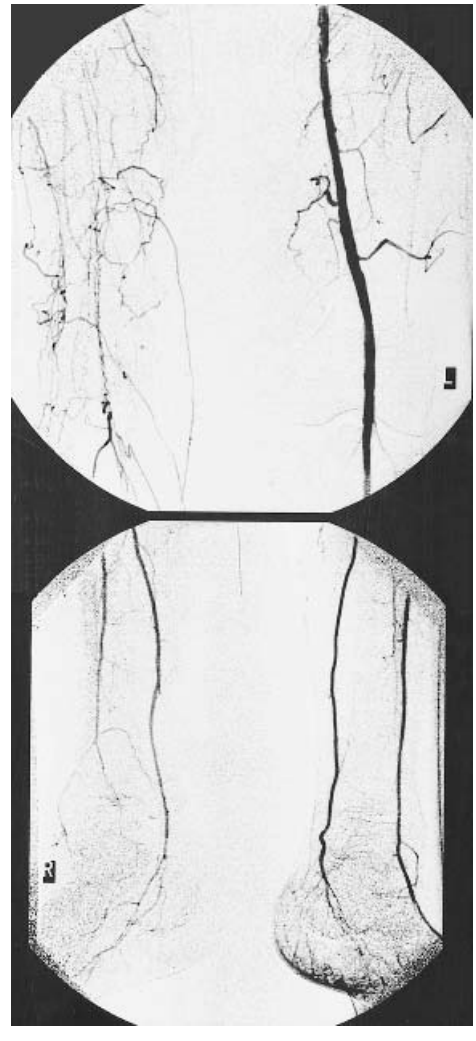

Figure 6.25 Arteriography of the patient whose foot is shown in Figure 6.23. There is severe obstruction of the distal part of the right femoral and popliteal arteries; the pedal arteries are patent and filled by collateral circulation

and netilmicin was added to the treatment regimen after the antibiogram.

An angiogram revealed severe obstruction of the lower right femoral and popliteal arteries (Figure 6.25). Vascular surgeons suggested a femoro-tibial bypass graft after his general condition had been stabilized for several months, or in the case of an emergency, since no gangrene was present at the time. Pentoxyphillin and buflomedil were prescribed. The ulcer improved after 2 weeks of antibiotic treatment and local care.

Keywords: Neuro-ischemic ulcer; angiography; osteomyelitis